

Epidemic dynamics

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2020-10-15

How does disease spread in a population? How do epidemics work?

Common processes

At this point we have all seen at least one epidemic, if not many, happen around us. There have been flare ups of measles in many communities with low vaccination rates, two large Ebola virus epidemics in X and Y in recent years, and of course the COVID-19 pandemic¹. And that is just epidemics in humans. In animals there are myriad epidemics every year, though most of the time these go unnoticed by the general population. Even plants have epidemic disease². While the host populations and the etiologies³ vary from viruses to bacteria to fungi to multicellular organisms⁴, disease ecologists and epidemiologists see a common set of processes at work in all of these epidemics.

1. Transmission. This is the process by which uninfected, but susceptible individuals become infected. There are several routes of transmission (e.g., by direct contact, from the environment, via the bite of some arthropod vector), but we'll come back to these later.
2. Virulence. This is the disease caused by infection—morbidity—potentially including death—mortality. This can range from barely detectable to mild (e.g., a fever and the sniffles) to very severe (e.g., bleeding out of every orifice). Actually, every biological and medical discipline seems to use this word differently⁵. I'm a disease ecologist and so we will think about virulence as the disease-induced reduction in host fitness. This usually implies direct mortality, although it could also be making an individual sick enough that they are more likely to die from other causes (e.g., predation, starvation). It would also include loss of reproductive output⁶.
3. Recovery. The job of the immune system is to prevent infections from taking hold and to clear them out if they get established. When we say recovery, we mean the latter. Yes, there's lots of fascinating complexities to how this all works⁷, but for our purposes we need to make only one distinction: Is there immune memory or not? If not, individuals recover, but can be re-infected. If there is memory, recovered individuals are resistant to re-infection, at least for some time⁸.

That's it. Sure, we can make all of these processes more nuanced and complex, but everything we might add, disease-wise, can be shoe-horned into one of these processes. Now let's see how they work together to make an epidemic!

¹ A pandemic is just an epidemic with a global scale.

² One disease ecologist has made a strong case that plants even have sexually transmitted infections. Pollinators, after all, often transmit infections.

³ Underlying causes of disease.

⁴ Parasitic barnacles? Check! Parasitic plants? There are many, including mistletoes. Birds? Yup! Just look at the brown cow birds.

⁵ To a virologist, virulence means the capacity to replicate efficiently in a host, but to a doctor virulence usually refers to the severity of symptoms.

⁶ There are many, many castrating parasites, and not all of them are sexually-transmitted.

⁷ It is worth noting that while we tend to take a very mammalian-focused view of immunology, with innate and acquired branches, B- and T-cells, cytokines and all of that, virtually every organism has some sort of immune system. Have you heard of the CRISPR-Cas9 system that is all the rage in molecular biology? It comes from the antiviral defense system of bacteria. And plants have sophisticated immune responses, too.

⁸ Yes, immunity to some pathogens wains. That is why you need booster shots, e.g., for tetanus.

A divided population

First, let us be explicit about our scale of interest. It is the population, some group of individuals that interact and are largely independent of other groups. Think of a city or a herd or all of the tadpoles in a pond. We are then interested in whether and how infections⁹ spread through this population, how many or what fraction¹⁰ are infected at any given time, how many or what fraction become infected, die, and so on. Given these goals, it makes sense that we need to divide the population into those that are susceptible, those that are infected, and, assuming that individuals can recover and become resistant to re-infection, a recovered/resistant group. We often call these groups *boxes* or *bins* or *compartments*¹¹ and for simplicity we call them S , I , and R .

The next step is to connect these boxes. How do we get new infections? They come from susceptible individuals that get infected through the process of transmission. What happens to the infected individuals? They might be lost from the population due to disease-induced mortality, a.k.a. virulence, or they can recover to the resistant class. That's it, at least for a simple infection in a simple population¹².

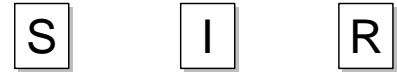
Now it is time to start thinking about the rates at which the three processes occur. That is, how big are those arrows? What influences whether they are big or small? Let's start with the easier ones, virulence and recovery.

Rates of flow from the I box

But first, let me emphasize the key word in that last paragraph: *rates*. Why are we focusing on say the rate of recovery rather than the probability or odds an individual recovers? The reason is that all of these processes, and the unfolding of the epidemic overall, are *dynamic*, they occur over time, and so how long individuals spend in one box or another matters. For instance, consider two infections with zero virulence and 100% recovery. Outcome-wise, they are identical, but what if the first infection was cleared in five days, on average, whereas the second one took 25 days. What would the size of the overall epidemics look like? Well, individuals with the second infection would be hanging around in the infectious box five times longer, meaning they'd have five times as long to pass on the infection. We would expect, all else equal, many more new infections from each infected individual, and much larger epidemics. Rates (and thus residence times) matter.

Now imagine we conduct an experiment where we infect animals with a pathogen of interest¹³ and then track how long they remain infectious. Let's say the animals remain infectious for ten days, on average. This is the infectious period¹⁴. We can calculate the rate at which individuals leave the I box, which we will call γ , as the inverse of the infectious period or $\gamma = 1/(10 \text{ days}) = 0.1$ per day.

But notice that we have two arrows leaving the I box. Animals can recover or die. Imagine in our experiment that 20% of the animals died and 80% recovered. We thus want to shunt 20% of the flow to death and 80% to the recov-

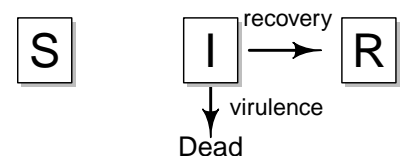


⁹ Remember, infection \neq disease.

¹⁰ Why do I keep saying number or fraction? Well, we can think about an epidemic in either way; the number (or density) of individuals that are sick, say, or the fraction or proportion of the entire population that are sick. They are *largely* interchangeable, but not always. I'll use numbers from here on out since it is more intuitive for most people.

¹¹ Indeed, these are called compartment models.

¹² Sure, we could add births and deaths due to other things. We could add carrying capacities. We could think about different age classes or sexes or those with and without pre-existing conditions. We could make this all kinds of complicated, but all of those are simply variations on this theme. And anyway, what's wrong with starting simply?!



¹³ Pick your favorite!

¹⁴ Assuming they become infectious immediately. More on this below.

ered/resistant box. Let us call the probability of death given an infection¹⁵, ϕ . Thus, the overall rate of disease-induced mortality, which we call virulence, is $\phi \times \gamma$. In our make-believe experiment this is $0.2/10 = 0.02$ per day.

Since those that do not die recover, we can write the recovery rate as $(1 - \phi) \times \gamma$.¹⁶ For our experimental data we would estimate the recovery rate as $0.8/10 = 0.08$ per day.

These are the *average* rates of disease induced mortality and recovery *for an individual*¹⁷. The overall flow of individuals out of the infected box and into, well, a heap on the ground¹⁸ is $0.02 \times I$ (or $\phi\gamma I$ more generally). Similarly, the flow of individuals out of the I box and into the R box is $0.08 \times I$ (or $(1 - \phi)\gamma I$).

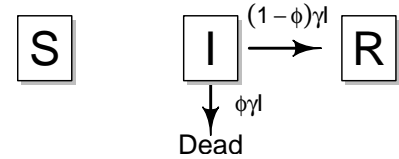
It is not essential that you are able to estimate virulence and recovery rates, but you should see that shorter residence times and larger proportions imply faster rates. It can also be an interesting exercise to think about what virulence might be for different sorts of infections, like influenza, which probably won't kill you, but if it does it does so quickly, and HIV/AIDS, which was very, very likely to kill you prior to effective treatments, but took years to do so. Which has higher virulence by our definition?

¹⁵ This probability, estimated by the proportion of infected individuals that die, is called the infection fatality rate. It's not a rate, but a probability or proportion, but whatever. It is similar to the *case* fatality (or mortality) rate, which is the proportion of individuals meeting a case definition that end up dying. Cases are always a subset of infections, so case fatality rates are generally higher than infection fatality rates.

¹⁶ Note that I am parameterizing these a bit differently than you are likely to see in most models. Most of the time we simply have a recovery rate, often called γ , and a virulence rate, often called α . The result is the same and I think this way is a bit clearer.

¹⁷ Like a *per capita* rate.

¹⁸ We haven't created a box for the dead individuals. Instead, we just know that animals leaving I to virulence are dead.



Transmission

So now we can describe what happens to the infected individuals in a population, but how do we get infections? This, the transmission term, is more complex, nuanced, and, I would argue, important than the other two. We'll start very simply, but beware, there be dragons ahead!

Let's first describe the steps that go into transmission, from the susceptible's point of view¹⁹.

- First a susceptible individual comes into contact with other individuals²⁰. at some rate, which we'll call $c(N)$ because I'm not feeling very creative. This way of writing it, though, suggests that the contact rate might be a function of population size, N , where $N = S + I + R$.
- Only a fraction of those individuals are infected: I/N .
- Then, given a contact with an infected individual, there is some probability, π , the susceptible individual becomes infected.

Putting this all together we get the rate at which a single susceptible individual is infected:

$$c(N) \times \frac{I}{N} \times \pi.$$

For simplicity, let's just assume that every susceptible makes c' contacts per day, like 10 or something. Importantly, we're focusing on *potentially infectious* contacts. This will depend on the pathogen or parasite. For some, just being in the same room or shaking hands might be sufficient, for others it might require prolonged close contact or sexual contact. Anyway, our rate per susceptible becomes

$$c' \frac{I}{N} \pi,$$

and if we say that $c' \pi = \beta$, since these are just two constant rates and so their product is a constant, and one term is simpler than two, we get

$$c' \pi \frac{I}{N} = \beta \frac{I}{N}.$$

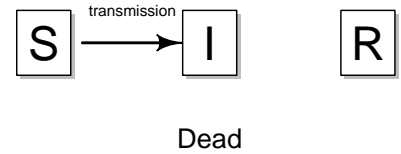
If $c' = 10$ potentially infectious contacts per day and each of those had a one in twenty chance of actually causing an infection, then $\pi = 1/20 = 0.05$ and $\beta = 10 \times 0.05 = 0.5$ per day.

Remember, this is the *per susceptible* rate of infection (or force of infection), and so the overall rate of infection (=transmission) is

$$\beta \frac{I}{N} S.$$

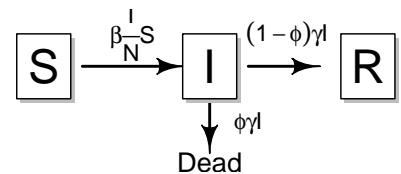
Putting it all together

We've now described the rates at which all three processes in an epidemic occur, in simple mathematical terms. I know that lowercase Greek letters can make people's



¹⁹ The rate at which an average susceptible individual becomes infected is called the force of infection (FOI) and it's pretty handy. It is sometimes possible to estimate the FOI from sentinels. For instance, sentinel chickens are routinely placed in various environments to estimate the rate at which they become infected with West Nile virus or other arboviruses.

²⁰ We'll focus on directly transmitted infections, but we could also replace hosts with, say, mosquitoes.



eyes glaze over²¹, but hopefully you followed along with the logic of how we thought about these rates. The next step is simply to put these all together in some equations that describe the rates or flows of individuals from one box to another.

Since there are three boxes, we'll have three equations, and each will describe the flows in and out of that box. So, starting with the susceptible box, and remembering that changes in the rate of some quantity, X , is described mathematically as dX/dt , we get:

$$\frac{dS}{dt} = -\beta \frac{I}{N} S.$$

Notice that there is no way to gain new susceptible individuals, since we are not including birth or recovery without resistance (or waning immunity). They only flow out as they become infected.

The infected box gains infections at this same rate of transmission as they are lost from susceptible class—this is a flow from S to I , after all—and then they are lost to virulence and recovery at a collective rate of $\phi\gamma I + (1 - \phi)\gamma I = \gamma I$.

$$\frac{dI}{dt} = +\beta \frac{I}{N} S - \gamma I.$$

Finally, we gain individuals in the recovered/resistant box as individuals recover, but there is no way to lose them since we are not thinking about waning immunity or other forms of mortality.

$$\frac{dR}{dt} = +(1 - \phi)\gamma I.$$

That's it! That's the whole model describing the dynamics of an epidemic of a directly transmitted infection with virulence and life-long immunity, absent any birth or non-disease death.

Now try something. Since we know that the whole population size is $N = S + I + R$, we can similarly add up all of the rates across all equations to get dN/dt . What are the dynamics of the whole population?

$$\begin{array}{r} \frac{dS}{dt} = -\beta \frac{I}{N} S \\ \frac{dI}{dt} = +\beta \frac{I}{N} S - \gamma I \\ \frac{dR}{dt} = +(1 - \phi)\gamma I \\ \hline \frac{dN}{dt} = \end{array}$$

A little aside on unit conversion

It is important and can be very helpful to remember that we have written out *equations*. The stuff on the right-hand side of the equal signs must equal the stuff on the left hand sides. This includes the units. So for instance, if dS/dt is in units of numbers of individual per time (e.g., days), everything on the right hand side

²¹ And uppercase Greek letters make people think of frats and sororities?

must, collectively, be in the same units of numbers of individual per time. So what are the units on β if $\beta \frac{I}{N} S$ has units of numbers of individuals per time? If I , N , and S are all numbers and we don't know what the units of β are we can write:

$$\frac{\text{numbers}}{\text{time}} = ? \times \frac{\text{numbers}}{\text{numbers}} \times \text{numbers}$$

We can see that the units of the fraction (the $\frac{I}{N}$) cancel each other out:

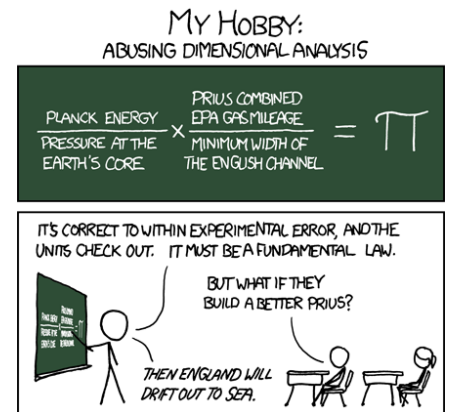
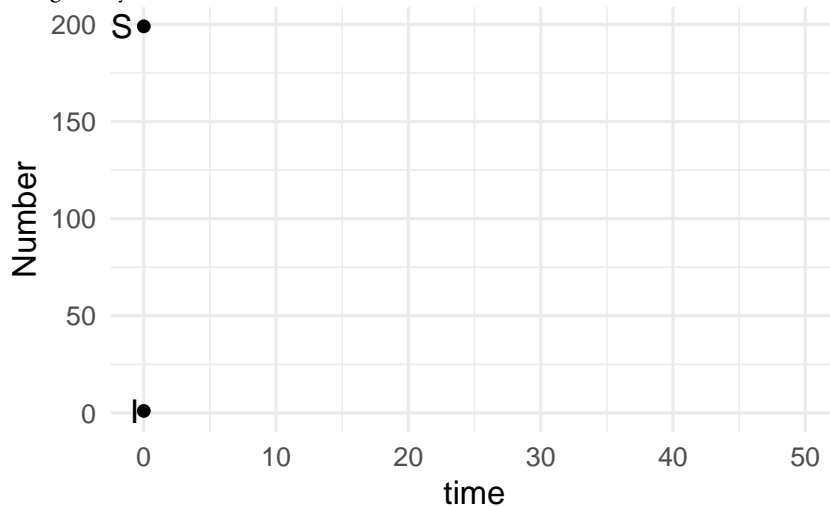
$$\begin{aligned} \frac{\text{numbers}}{\text{time}} &= ? \times \frac{\cancel{\text{numbers}}}{\cancel{\text{numbers}}} \times \text{numbers} \\ \frac{\text{numbers}}{\text{time}} &= ? \times \text{numbers} \\ ? &= \frac{1}{\text{time}} \end{aligned}$$

This means that β must have units of *per time*. Try it!

This can be a useful way of making sure you did things correctly and also that you are thinking about your rates correctly. But do beware: just because your units work out does not mean you have the right answer!

The dynamics of an epidemic

So we have our model, but, I can almost hear you asking, what does it *do*? We're about to get there, simulating or running or solving these equations²² so that we can see how S , I , and R change through time. But first I want you to take a moment and draw on these axes what *you* think these three quantities will look like through time. Try it. Start at time zero with a large susceptible population (S is big), a single infected ($I = 1$), and no resistant individuals ($R = 0$). What happens to these three groups as the epidemic progresses? You would probably do well to move in small steps, starting with S and then thinking about I and R . Try drawing the dynamics on these axes.



²² We won't worry about these details of how, but if you're interested, it's a version of the Runge-Kutta method. Or, like I said, don't worry about it.

OK, now here's what the model actually does (using the parameters we imagined above). How does it differ from your predictions? Why? (This is a good time to stop and refine or reconcile your thinking before moving on.) Do you understand why I goes up and down? Why S only declines and R only increases? What happened to the total population size, N ? Does this make sense to you?

You'll probably notice that the model describes fractions of individuals in each box. This is because these equations describe numbers continuously, not as discrete individuals. You can make a model with discrete individuals, but it will necessarily be stochastic, involving chance events (e.g., did individual 99 get infected in this time step or not?). These ordinary differential equations (ODEs) are best thought of as describing the *average* dynamics and work best in large populations.

Another aside: including an incubation period

You may have noticed or recall that we are assuming that all individuals in the I class are equally infectious. One consequence of this is that infected individuals are *instantaneously* infectious to others. But most infections take a bit to ramp up; there is an incubation period²³. To account for this we can include an exposed-but-not-yet-infectious class, E . Newly infected individuals flow into the E box with transmission and then flow out at a rate $k = 1/(\text{the incubation period})$, on average. Everything else is the same.

Indeed, the dynamics are usually very similar from an SEIR to an SIR. The delay, well, delays things a bit and so the steepness of the curves will vary. The shorter the incubation period, the more like an SIR it will become until at some point you might as well use an SIR.

This is just one example of how you can modify a compartment model to better match the biological realities of your system. We will see more later.

Why epidemics fade: think of fire

Lastly, why did the epidemic fade? That is, why did the infected category eventually decline to near zero levels? I hope you can intuitively answer that: like a fire running out of fuel, the epidemic ran out of susceptible individual to infect (around day 12 or 13) and then those individuals that were infected at that point eventually died or recovered.

We can better understand the growth and decline of those infected by thinking about the rate at which infections are gained (and lost) as a function of the number of infected individuals, I , sort of like we did when thinking about population growth rate in the exponential and logistic models.

$$\frac{dI}{dt} = \overbrace{\beta \frac{I}{N} S}^{\text{gains}} - \overbrace{\gamma I}^{\text{losses}}$$

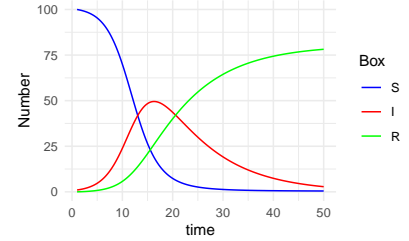
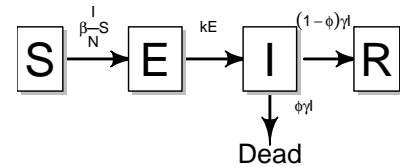


Figure 1: Epidemic dynamics of an SIR model with $\beta = 0.5$, $\gamma = 0.1$, and $\phi = 0.2$ and starting from a population with $S = 100$ and $I = 1$.



²³ Doctors tend to define the incubation period as the time until symptoms develop, but epidemiological modelers would usually think of it as the time until they are infectious. They *can* be the same thing, but not always.

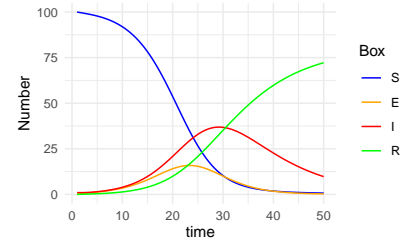


Figure 2: Epidemic dynamics of an SEIR model with $\beta = 0.5$, $k = 0.33$, $\gamma = 0.1$, and $\phi = 0.2$ and starting from an a population with $S = 100$ and $I = 1$.

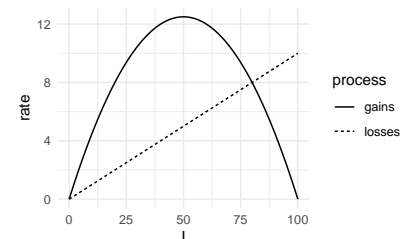


Figure 3: Rates of gains (solid line) and losses (dotted line) as a function of I with $\beta = 1$, $\gamma = 0.1$, and, for simplicity, $\phi = 1$.

We can see that the rate of losses increases linearly with I , with a slope of γ , but it will take a bit more to understand the gains term, or transmission. First, let us simplify things a bit and ignore the recovered group, R , by setting $\phi = 1$, meaning everyone dies. Since we can ignore the R class, we see that $N = S + I$ and, by rearrangement, $S = N - I$. If we place this in the gains equation and do some re-arranging we get:

$$\begin{aligned} \text{gains} &= \beta \frac{I}{N} S \\ &= \beta \frac{I}{N} (N - I) \\ &= \frac{\beta}{N} \underbrace{I}_{\text{source}} \underbrace{(N - I)}_{\text{recipient}} \end{aligned}$$

You can see that the overall rate of transmission is a function of the source of infection, I , and the recipients, $S = N - I$. Too few of one or the other and the overall rate of transmission is reduced.

Alternatively, we can multiply through and separate terms

$$\begin{aligned} \text{gains} &= \beta \frac{(N - I)}{N} I \\ &= \beta \frac{IN}{N} - \beta \frac{I^2}{N} \\ &= \beta \frac{N}{N} I - \frac{\beta}{N} I^2 \\ &= \beta I - \frac{\beta}{N} I^2 \end{aligned}$$

to get an equation for a parabola. At low values of I the second, negative terms is small and barely matters, but as I gets big it becomes more and more important and starts to dominate.

So, both mathematically and, I hope, intuitively we can see why epidemics fade: they run out of susceptibles. (This also hints at how we end up with endemic or persistent infections in populations. But more on this later.)

What does it take for an epidemic to grow? (R_0)

Thinking of the equation for I in terms of gains and losses also provides some insights into what it takes for an epidemic to grow. Intuitively, if the epidemic is to grow, the equation for $\frac{dI}{dt}$ must be positive. This only occurs if the gains are greater than the losses, or $\beta \frac{I}{N} S > (\alpha + \gamma)I$.

$$\frac{dI}{dt} > 0 \text{ only if } \overbrace{\beta \frac{I}{N} S}^{\text{gains}} > \overbrace{(\alpha + \gamma)I}^{\text{losses}}$$

We can also think about this from the point of view of a single infected individual entering an otherwise wholly susceptible population (i.e., $I = 1$ and $S = N - 1 \approx N$). How many new cases will they produce over the length of their infection? If

it is greater than one the epidemic will grow, but if it is less than one the infection will not spread and the epidemic will fade out before it gets going²⁴. So what is the rate at which the single I causes new cases? That is simply $\beta \frac{S}{N}$ (remember, we said $I = 1$, so it disappears from the gain term), which is essentially β since we said that $S \approx N$.

Next, how long, on average, does a single infected individual stay infected before recovering or dying? Well, the average duration an individual spends in a box, such as the I box, is one over the rate at which it leaves the box—a faster rate of loss means a shorter residence time. In this case, the rate of loss is γ (whether they go to the R box or the heap of dead on the floor) and so the average residence time or life time of an infection is $1/\gamma$.

We can then put these two together to get the average number of new, secondary cases by a single infected individual over the average life time of its infection²⁵:

$$\begin{aligned} R_0 &= \beta \times 1/(\gamma) \\ &= \frac{\beta}{\gamma}. \end{aligned}$$

This is the magic term R_0 , often called “R-naught” from the British English habit of calling zero, naught. It is also known as the reproductive number of the infection²⁶. If $R_0 > 1$, meaning that an infection is expected to more than replace itself, the epidemic is expected to grow, on average, and if $R_0 < 1$, meaning an infection will not replace itself, the epidemic is expected to fade before it really gets going.

For context, one study²⁷ of early estimates of R_0 for COVID-19 found values from a low of 1.5 to a high of 6.68 and one²⁸ from the Diamond Princess cruise ship, which was a nice, closed and well-studied population, was 2.28. All of these estimates suggest that COVID-19 would tend to grow, but by different amounts. Why the range of estimates? Some of it is just a product of statistical uncertainty and data sets of varying size and quality. But more fundamentally, that rates of transmission and the duration of infection are going to depend on the population in which the infection is spreading (e.g., young or old, healthy or sick) and the local conditions (e.g., crowded or not, little or lots of movement and contacts, as well as, perhaps, temperature and humidity). In other words, R_0 is *not* a fixed characteristic of an infection or pathogen, but rather a product of that pathogen in a particular host population.

It is important to remember that R_0 is defined with a single infected entering a wholly susceptible population. When an epidemic has begun this is no longer the case and so we instead think of the *effective* reproductive number, R or R_t , which describes the same things given whatever I and S are at the time²⁹.

An aside on estimating R_0

You may recall that when we were discussing processes that grow exponentially, we included the early dynamics of epidemics. We also noted that log of a popu-

²⁴ Importantly, we are thinking of this *on average*. When dealing with real individuals in real populations in, well, reality, chance events can be very important. If you introduced of a single infected individual into a whole bunch of identical susceptible populations you would get a whole range of outcomes, but on average the results would look like our calculations.

²⁵ You can get to the same place, at least in these simple models, by describing R_0 as the ratio of gains to losses in the dN/dt equation, again assuming that $I = 1$ and $S \approx N$:

$$\begin{aligned} R_0 &= \frac{\text{gains}}{\text{losses}} \\ &= \frac{\beta \frac{1}{N} N}{\gamma} \\ &= \frac{\beta}{\gamma} \end{aligned}$$

²⁶ And its interpretation is analogous to the reproductive number of an organism estimated from a life table, $R_0 = \sum l_x b_x$ if you’ve seen one elsewhere.

²⁷ Liu et al. (2020) J. Travel Med.

²⁸ Zhang et al. (2020) Int. J. Infect. Dis.

²⁹ See this site for state-by-state estimates of R_t , <https://rt.live/>

lation ($N(t)$, whether rice grains or infections) growing exponentially over time would follow a straight line with a slope of r , which we called the intrinsic rate of increase.

$$\log(N(t)) = \log(N_0) + r \times t$$

Well, let's see what happens if we plot our simulated epidemic on a logarithmic y-axis.

Clearly, none of these lines are straight over the whole time period, but look at the line for the infected individuals. It looks like its initial increase is following a straight line for a while, and then it seems to decline along a straight line later. (You can probably find some bits of the other two lines that approximately straight for some stretches, too. But let us focus on the I box.) Let me zoom in on the first ten days of the I line and fit a linear regression to it:

It looks like pretty close, but not perfect fit. If you look very, very closely you will see that the red line curves downwards a bit, so the regression is a bit too flat early on and a bit too steep later on. But it is not a bad approximation. The slope of this linear regression on a natural log scale is 0.387. In other words, this slope is approximating r . But we want R_0 . A simple extrapolation, from a rather more complex analysis³⁰ is:

$$R_0 \approx 1 + r/\gamma,$$

from which we get $1 + 0.387/0.1 = 4.871$. This is a pretty close estimate of the “known” value of $R_0 = \beta/\gamma = 0.5/0.1 = 5$.

In actual practice, researchers *do* use this approach to estimate R_0 , but actually sorting out the duration of time in the infected class can be tricky and nuanced. There are many other more sophisticated approaches to estimating R_0 , as you might expect, but it is nice to see that 1) simple can work and 2) that, once again, plotting numbers against time on a logarithmic scale tells us something about the underlying process³¹.

What does it take for an epidemic *not* to grow? Reducing $R_t < 1$

You can think about most public health interventions as aimed at reducing R_t or R_0 so that, on average, each infected individual produces less than one new infection. When this happens, the epidemic will start to fade. Of course this happens naturally when an infection has spread so much that it runs out of susceptible hosts³². Like a wildfire running out of unburned trees, the spread of the infection will slow down and eventually fade out. What's more, like the unburned trees after a fire, an epidemic will not generally infect everyone, even if left uncontrolled. This was one of the more surprising results from early epidemic models. Even highly transmissible infections will not reach everyone.

This finding, and the underlying logic, that we might reach a critical level of “heard immunity”, has featured prominently in discussions about how the COVID-19 pandemic might end. If a large enough fraction of the population is immune (i.e., in the R class) then infected individuals will, on average, fail to pass

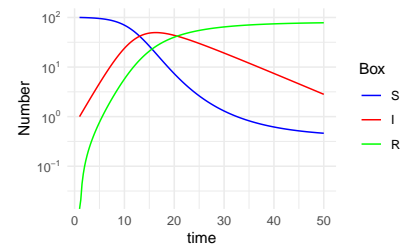


Figure 4: Epidemic dynamics of an SIR model on a logarithmic y-axis. The parameters and results are the same as above.

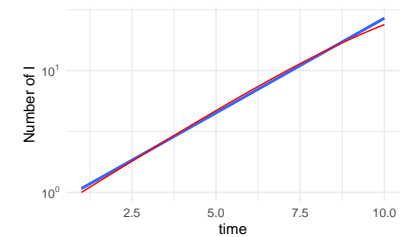


Figure 5: The first ten days of dynamics of the infected box from an SIR model on a logarithmic y-axis. The blue line is from a linear regression.

³⁰ Wallinga & Lipsitch (2007) *Proc. B Roy. Soc.*

³¹ I'll let you think about what the linear *decline* in I on the graph above means and what the slope might represent.

³² Although it can take a long time get there!

on their infection to someone else and the epidemic will wain. This has sometimes led to a I-might-as-well-get-it-over-with attitude in some, although [this is not without serious risks](#). A much better strategy is to intervene earlier in ways that reduce R_t or $R_0 < 1$.) Indeed you can map all interventions on to one or more components of the equation for R_0 or the underlying bits in our SIR model.

- Vaccination essentially reduces S and puts those individuals into the R class. This means fewer individuals that can get sick and, if you get a high enough fraction of the population vaccinated, that first infected individual will not find enough susceptibles in the population to replace itself before it dies or recovers. This is herd immunity, but without all of the morbidity and mortality to get there³³. What's more, you can protect particularly vulnerable groups, such as the very young or old or immunocompromised, by vaccinating the people around them.
There is a simple equation that describes the fraction of the population that must be vaccinated, which we call q , to ensure that $R_0 \leq 1$: $q \geq 1 - \frac{1}{R_0}$. As you would expect, the higher R_0 , the greater that fraction you must vaccinate.
- Quarantine and social distancing and masks are all aimed at reducing the number of potentially infectious contacts (remember $c(N)$?) or the probability of transmission given a contact (π), all of which should reduce β . From a public health point of view it does not matter whether a mask protects the wearer from infection or other people from an infected wearer, both reduce π .
- Contact tracing is especially important because it allows one to focus these efforts to reduce infectious contacts on 1) the infected people and 2) their close contacts that might have been infected. With good contact tracing³⁴ one need not apply control measures blindly across a whole population. Indeed, most successful control programs, such as the recent end to the Ebola virus outbreak in northeastern [Democratic Republic of the Congo](#), involve contact tracing in combination with quarantine and, often, targeted vaccination.
- Treatment of infections, often with chemical therapeutics, can reduce the duration of an infection, as well as reduce morbidity and mortality. If individuals are infectious for less time³⁵, there is a shorter window for transmission, which can reduce R_0 .

If you would like to try your hand at ending the COVID-19 epidemic, try this fun interactive application: <https://alhill.shinyapps.io/COVID19seir/>. It includes a lot of sliders, but the essential features of the system (outside of space and stochastic dynamics) are there.

³³ Yes, vaccines can have side effects, but those are much, much, much less severe than the disease.

³⁴ Which requires good testing broadly applied.

³⁵ It is important to note that some treatments and vaccines can reduce symptoms in a person or animal without reducing their ability to spread the infection. This is good for the individuals, sure, but it can exacerbate the spread of infection, making the epidemic worse. This is one of many potential conflicts between what is optimal for an individual vs for a population.